cream only. FIG. **15** also show that the NAD content of skin removed from the abdomen of the treated animal was not increased, providing proof that the increased NAD content was due to topical delivery.

In another experiment, the effect of the number of daily application of 1% tetradecylnicotinate cream was examined. Again, application was made to the back of the animal and the abdomen of each animal served as control. The results, depicted in FIG. **16**, show that the NAD content of skin removed from the back increased as a function of the number of daily applications. In contrast, the NAD content of the abdomen did not increase, providing additional evidence that the increased NAD content was due to topical delivery of the pro-NAD agent.

It should be noted that in no case were any signs of toxicity observed as a result of the topical application of tetradecylnicotinate cream in the experiments shown in FIGS. **15** and **16**.

Other pro-NAD agents may be identified by exposing cells in culture to candidate pro-NAD agents or by exposing skin on a test subject, such as a mouse, to the candidate pro-NAD agent. After a safe and effective dosage is determined, the candidate pro-NAD agents may be tested on human volunteers and assayed by skin biopsy samples. The effectiveness of the pro-NAD agents may be determined by (a) biochemically analyzing cell lysates to assess the cellular NAD content or (b) scoring phenotypic or functional changes in treated cells as compared to control cells that were not exposed to the candidate pro-NAD agent.

Where analogs and derivatives of a known pro-NAD agent are to be identified or evaluated, the cells are exposed to the pro-NAD agent of the invention and compared to positive controls which are exposed only to the known pro-NAD agent, and to negative controls which were not exposed to either the candidate pro-NAD agent or the known pro-NAD compound.

In order to determine if the pro-NAD agent administered according to the method of the invention is absorbed into body tissues, and if so, in which tissue absorption occurs, the following may be performed. Samples of various body tissues from a subject, such as a laboratory mouse, were analyzed for NAD content at increasing hours after oral administration of a pro-NAD agent. The results of the measurement are compared to that of control subjects to determine the percent increase of NAD content. A dose response curve and a therapeutic index can be developed to determine the optimal oral dosage.

Other embodiments and uses of the invention will be apparent to those skilled in the art from consideration of the specification and practice of the invention disclosed herein. All U.S. patents and applications and other references noted herein are specifically incorporated by reference. The specification and examples should be considered exemplary only with the true scope and spirit of the invention indicated by the following claims.

We claim:

1. A method for treating or slowing skin deterioration in a subject in need thereof, comprising administering to said subject a composition consisting essentially of a nicotinic acid alkyl ester, wherein the alkyl moiety of said nicotinic acid ester is a straight alkyl chain of from 12 to 16 carbon atoms, in an amount sufficient to increase intracellular NAD content in skin cells of said subject, and to treat or to slow skin deterioration thereby.

2. The method of claim **1**, comprising administering said composition topically, intradermally, subcutaneously, via dermal patch, orally, parenterally, or via slow release mechanism.

3. The method of claim **1**, wherein said alkyl chain contains 14 carbon atoms.

4. The method of claim **1**, wherein said skin deterioration is caused by DNA damage.

5. The method of claim **1**, wherein said skin deterioration is caused by ultraviolet damage.

6. The method of claim **4**, comprising administering said nicotinic acid alkyl ester in an amount sufficient to elevate an intracellular skin protein, selected from the group consisting of PARP-1, PARP-2, PARP-3, tankyrase, V-PARP, and polymerase.

7. The method of claim **5**, comprising administering said nicotinic acid alkyl ester prior to exposure to ultraviolet radiation.

8. The method of claim **5**, comprising administering said nicotinic acid alkyl ester after exposure to ultraviolet radiation.

9. The method of claim **1**, wherein said subject is a mammal.

10. The method of claim **8**, wherein said mammal is a human.

11. A method for treating or slowing skin deterioration in a subject in need thereof, comprising administering a composition to said subject which comprises a nicotinic acid alkyl ester, wherein the alkyl moiety of said nicotinic acid alkyl ester is a straight chain alkyl of from 10 to 22 carbon atoms, and further comprises at least one functional group selected from the group consisting of thiol, alcohol, amine, caboxylic acid, oninum, carboxylic anhydride, carboxylic ester, acylhalide, amide, nitrile, aldehyde, ketone, imine, ether, sulfide, halcide, nitro, nitroso, azide and drazo group, in an amount sufficient to increase intracellular NAD content in skin cells of said subject and to treat or to slow skin deterioration thereby.

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